A STUDY OF LIPID PROFILE IN CHRONIC KIDNEY DISEASE PATIENTS

DISSERTATION

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BONAFIDE CERTIFICATE

Certified that this dissertation is the bonafide work of ***Dr.A.VINOTH*** *on* "**A STUDY OF LIPID PROFILE IN CHRONIC KIDNEY DISEASE PATIENTS**" *during his M.D. (General Medicine) course from May 2006 to March 2009 at the Madras Medical College and Government General Hospital, Chennai.*

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DECLARATION

I solemnly declare that the dissertation titled ***"A STUDY OF LIPID PROFILE IN CHRONIC KIDNEY DISEASE PATIENTS"*** *is done by me at Madras Medical College & Govt. General Hospital, Chennai during 2007-2008 under the guidance and supervision of* ***Prof. R.SUKUMAR, M.D.,***

This dissertation is submitted to The Tamil Nadu Dr.M.G.R. Medical University towards the partial fulfillment of requirements for the award of M.D. Degree (Branch I) in General Medicine.

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CONTENTS

INTRODUCTION

Hyperlipidemia, one of the important risk factor of atherosclerosis, is an abnormality commonly encountered in patients with chronic kidney disease. The increased risk of atherosclerotic cardiovascular disease may be due to hyperlipidemia. Other risk factors predisposing to cardiovascular disease in chronic kidney disease patients include diabetes, hypertension, obesity and smoking.

Dyslipoproteinemia is an additional risk factor for the progression of renal insufficiency. It has been shown in a large population of patients with chronic kidney disease that the rate of progression was significantly higher in hyperlipidemic patients compared with normolipidemic patients[1].

The pathogenesis of chronic allograft dysfunction is complex and results from various factors. Among them hyperlipidemia is an important factor implicated in the development and progression of chronic allograft dysfunctions. In an observational study, it was found that hypertriglyceridemia and the Lp (a) >30mg/dl before and

after transplantation were independent risk factors for chronic allograft dysfunction[2].

Factors such as race, gender, age and diabetic status potentially confound the interpretation of the lipoprotein profile[3].

Indian studies on lipid abnormalities in chronic kidney disease have not been consistent. **Sharma et al.,**[4] **Kunde et al.,**[5] found no hyperlipidemia whereas **Gupta et al.,**[6] **Das et al.,**[7] observed hypertriglyceridemia and reduced HDL levels in CKD patients as in western studies. In view of inconsistency and limited evidence in southern part of this country it was decided to study the lipid profile in our patients with chronic kidney disease.

AIMS OF THE STUDY

1. To estimate various lipid profile abnormalities in Chronic Kidney Disease patients.

2. To identify the predominant lipid pattern in chronic kidney disease patients.

3. To study the correlation between the serum creatinine levels and lipid abnormalities in Chronic Kidney Disease.

4. To estimate the prevalence of Left Vetricular Hypertrophy and Ischemic Changes in patients with chronic kidney disease.

REVIEW OF LITERATURE

CHRONIC KIDNEY DISEASE DEFINITION:[8,9]

K/DOQI - Kidney Disease Outcome Quality Initiative definition of CKD is

1. Kidney damage for ≥3 months is defined by structural or functional abnormalities of the kidney with or without reduction in GFR manifest either by
   a. pathological abnormalities or
   b. Markers of kidney damage including abnormalities of composition of blood or urine or abnormalities in imaging tests.
2. GFR <60ml/min/1.73m^2 for ≥3 months with or without kidney damage.

HISTORY:

The word “Uremia” is coined by Piorry & L.Heritier in 1840. Association between lipid abnormalities and pathogenesis of renal disease was first suggested by virchow in 1860. He described extensive fatty degeneration in autopsy tissue from patients with Bright’s disease.

EPIDEMIOLOGY:

In India, with a population of one billion and an estimated incidence of ESRD of 100 per Million Population, approximately 100,000 patients develop ESRD each year. Of these 90% never see a nephrologist. Of the 10,000 patients who do consult a nephrologist renal replacement therapy started in 90%; the other 10% are unable to afford any form of renal replacement therapy[10].

The majority of the 9000 patients who receive renal replacement therapy are begun on hemodialysis. Of the 8500 patients, who are on hemodialysis, about 60% lost their follow-up within 3 months. Approximately 9-13% of patients die within one year while on treatment[11,12].

About 17-23% of patients undergoing renal transplantation should be on dialysis for 2-3 months for pre-transplant stabilisation. Although 4% of patients remain on maintenance HD, very few stay on maintenance HD longer than 24 months[11,12].

Patients mobilise the resources for the expenditure from the following[13]:

1. 4% - Pooled family resources
2. 60% - Employer
3. 20% - Selling property and jewels
4. 20% - Loans

ETIOLOG Y

Common causes of ESRD in India are

Disease %	***Center A***[12]	***Center B***[14]	***Center C***[15]
CGN (Chronic glomerulo nephritis)	28.6	36.64	18.20
Diabetic nephropathy	23.2	23.84	26.76
Chronic interstitial nephritis	16.5	14.35	21.05
Hypertensive nephrosclerosis	4.1	13.4	10.06
Obstructive nephropathy	6.4	–	1.22
Adult polycystic kidney disease	2.0	3.53	2.07
Unknown	16.2	3.76	–

In developing countries like India, majority of patients die without receiving any form of dialysis. The number of patients accepted by dialysis programs is about

80 pmp - Egypt[16]

20 pmp - Malaysia[17]

3-5 pmp - India & China[18]

PATHOPHYSIOLOGY OF CHRONIC KIDNEY DISEASE[19]

The pathophysiology of CKD involves two broad sets of mechanisms of damage (1) Initiating mechanisms specific to underlying etiology (Immune complexes and mediators of inflammation in certain type of glouerulonephritis, or toxin exposure in certain diseases of the renal tubules and interstitium) and 2) a set of progressive mechanisms involving hyperfiltration and hypertrophy of the remaining viable nephrons, that are a common consequence following long term reduction of renal mass, irrespective of underlying etiology. The responses to reduction in nephron number are mediated by vasoactive hormones, cytokines, and growth factors. Eventually, these short term adaptations of hypertrophy and hyperfiltration become maladaptive as the increased pressure and flow predisposes to sclerosis and dropout of remaining nephrons. Increased intrarenal activity of the renin-angiotensin axis appears to contribute both to initial adaptive hyperfiltration and to the subsequent maladaptive hypertrophy and sclerosis, the latter, in part, owing to the stimulation of transforming growth factor β;(TGF β). This process explains why a reduction in renal mass from an isolated insult may lead to a

progressive decline in renal function over many years.

Stages of CKD[19]

National Kidney Foundation (KDOQI) laid guidelines for the definition of stage of CKD.

Stages	***GFR, ml/min per 1.73m^2***
0	>90[a]
1	≥90[b]
2	60-89
3	30-59
4	16-29
5	<15

a) With risk factors for CKD (HTN, DM, autoimmune disease, old age, African ancestry, family history, H/o ARF, Proteinuria, abnormal urinary sediment, abnormal urinary test)

b) With demonstrated kidney damage e.g. persistent proteinuria, abnormal urinary sediment, abnormal blood and urine chemistry, abnormal imaging studies.

CLINICAL FEATURES:

Fluid and electrolyte disturbances

- Volume expansion
- Hyponatremia
- Hyperkalemia
- Hyperphosphatemia

Endocrine - Metabolic disturbances

- Secondary hyperparathyroidism
- Adynamic bone disease
- Vit. D deficient osteomalacia
- Carbohydrate resistance
- Hyperuricemia
- Hypertriglyceridemia
- Increased Lp (a) levels
- Decreased high density lipoprotein level
- Malnutrition
- Amenorrhea ,infertility and sexual dysfunction
- β_2 microglobulin associated amyloidosis

Neuromuscular Disturbances

- Fatigue
- Sleep disorders
- Headache
- Impaired mentation
- Lethargy
- Asterixis
- Muscular rigidity
- Peripheral neuropathy
- Restless leg syndrome
- Myoclonus
- Seizures
- Coma
- Muscle cramps
- Dialysis disequilibrium syndrome
- Myopathy

Cardiovascular and pulmonary complication

- Arterial hypertension
- Congestive heart failure or pulmonary edema
- Pericarditis
- Hypertropic or dilated cardiomyopathy
- Uremic lung
- Accelerated atherosclerosis
- Hypotension and arrythmias
- Vascular calcification

Dermatologic Disturbances

- Pallor
- Hyperpigmentation
- Pruritus
- Ecchymoses
- Fibrosing dermopathy
- Uremic frost

Gastro Intestinal disturbances

- Anorexia
- Nausea and vomiting
- Gastroenteritis
- Peptic ulcer
- Gastrointestinal bleeding
- Idiopathic ascites
- Peritonitis

Hematologic and Immunologic disturbances

- Anemia
- Lymphocytopenia
- Bleeding diathesis
- Increased susceptibility to infection
- Leukopenia
- Thrombocytopenia

Treatment

The optimal timing of therapy is usually well before a measurable decline in GFR and certainly before CKD is

established.

Clinical Action Plan[19]

	GFR ml/min/ $1.73m^2$	Action [a]
Kidney damage with normal or increased GFR	≥90	Diagnosis and treatment, treatment of comorbid conditions, slowing progression, CVD risk reduction
Kidney damage with mild decrease in GFR	60-89	Estimating progression
Moderate ↓ GFR	30-59	Evaluating and treating complications
Severe ↓ GFR	15-29	Preparation for kidney replacement therapy
Kidney Failure	<15 (or dialysis)	Kidney replacement (if uremia present)

[a]-includes actions from preceding stages.

Renal replacement therapy includes both dialysis and transplantation.

Slowing the progression of renal disease in CKD can be accomplished by the following.

1. Diet - Protein restriction 0.6-0.75g/kg/day

   - Low salt 60-80mmol/day

2. Blood pressure control - Bp <130-135/80-85mmHg

   if proteinuria <1g/24 hr

   Bp <125/75 mmHg if proteinuria >1g/24 hr

3. Proteinuria –to reduce to <1g/24hr

   use an ACE inhibitor or angiotensin receptor antagonist

4. Glycemic control in DM - Hb A_{IC} < 7%

5. Dyslipidemia -Control individual lipid fractions

6. Smoking - cessation

7. Alcohol - Restriction to less than 2 drinks per day.

DYSLIPIDEMIA:

Dyslipidemia is empirically defined as plasma lipids that are associated with adverse outcomes such as cardiovascular disease[20].

Normal structure and fuction of lipoprotein:

Lipoproteins & Apolipoproteins:

- Lipoproteins consist of lipids and proteins known as apolipoproteins (apo) with the main function of

transporting water insoluble lipids such as cholesterol or triglycerides in plasma, from sites of absorption (gut) and/or synthesis (liver) to the sites of utilization (peripheral tissues) or processing.

- In addition to their role in the formation of lipoproteins apolipoproteins perform a variety of functions in the metabolic conversions of lipoproteins including secretion, retardation of premature removal, recognition of binding & removal sites and activation of lipolytic enzymes.

Traditionally, lipoproteins are classified on the basis of their density properties[21]

1. Chylomicrons (<0.94g/ml density)
2. Very low density (0.94 - 1.006g/ml density)
3. Intermediate density (1.006 - 1.019 g/ml density)
4. Low density (1.019 - 1.063 g/ml density)
5. High Density (1.063 - 1.21 g/ml density)

LIPOPROTEIN PATHWAYS:

Lipoprotein pathways are divided into exogenous pathway and endogeneous pathway.

EXOGENOUS PATHWAY:

In this pathway chylomicrons transport dietary lipids that are absorbed from the intestine via the systemic circulation. Chylomicrons are triglyceride rich and normally catabolized within minutes by the endothelium - associated lipoprotein lipase (LPL), thereby generating free fatty acids (FFA), which are taken up by the liver, muscle and adipose tissues. During this catabolic process, chylomicrons diminish in size and become chylomicron remnants, which are taken up by the liver via the low-density lipoprotein (LDL) receptor and the LDL receptor - related protein (LRP).

ENDOGENOUS PATHWAY [22]

In this pathway, the liver assembles and secretes triglyceride - rich VLDL particles, which transport triglycerides from the liver to peripheral tissues. After hydrolysis of the triglycerides by LPL (Lipoprotein lipase) the VLDL particles are reduced to intermediate density lipoproteins which can be taken up by the liver or can be further hydrolysed to LDL particles. During this conversion, the particles become depleted of triglycerides but retain considerable amounts of cholesterol .

LDL transports cholesterol primarily to hepatocytes but also

to peripheral tissues. APO B-100 is responsible for the recognition and uptake of LDL by the LDL receptor, which clears approximately 60-80% of LDL in normal individuals. The remaining LDL is removed by other specific receptors such as LRP or by scavenger receptors[23].

Oxidized LDL (OX-LDL) in particular can be taken up by scavenger receptors on macrophages and vascular smooth muscle cells. When these macrophages become overloaded with cholesterol esters, they transform into foam cells, which is a major step in the development of atherosclerosis. When LDL becomes lipid depleted small dense LDL (sd LDL) is generated which has lower affinity for the LDL receptor but is more susceptible to oxidative modification. Thus sd LDL is more atherogenic than larger LDL particles[24].

High-density lipoprotein plays an important role in reverse cholesterol transport, which shuttles cholesterol from peripheral cells to the liver an important step that relieves the peripheral cells from cholesterol burden[25]. HDL precursor particles are secreted by the liver and intestine and can absorb free cholesterol from cell membranes; a process that is mediated by ATP binding

cassette transporter-1, apoA-I, apoA-IV. ApoA-I is the major apolipoprotein of HDL and activates lecithin: Cholesterol acyltransferase which esterifies the accepted free cholesterol for transport.

By acquisition of additional apolipoproteins, cholesterol esters and triglycerides, HDL3 particles are transformed into larger HDL2 particles[26]. Reverse cholesterol transport can take three different routes. First large HDL particles with multiple copies of Apo E can be taken up by the liver via the LDL receptor. Second, the accumulated cholesterol ester from HDL can be selectively taken up by the liver mediated by scavenger receptor B1[27]. This receptor is expressed primarily in liver and nonplacental steroidogenic tissues. Third, cholesterol esters are transferred by the cholesteryl ester transfer protein from HDL to triglyceride rich lipoproteins. Plasma HDL cholesterol levels are influenced by the complexity of these reverse cholesterol transport process. Disturbances in the concentration of apoproteins, function of enzymes, transport proteins, receptors, other lipoproteins and the clearance from plasma can have a major impact on the anti atherogenic properties of HDL.

***Pathophysiology of dyslipidemia in CKD*[28-31]**

Plasma triglycerides are predominantly found in two types of lipoproteins in normal individuals. These are chylomicrons which are assembled in the intestine for the transport of dietary fatty acids, and VLDL, which are produced in the liver for the transport of endogenous fatty acids[32-34].

Elevated triglycerides are the consequence of both high production rate and a low fractional catabolic rate[35].

Increased production of triglyceride rich lipoproteins is possibly due to

1. Impaired carbohydrate tolerance.
2. Enhanced hepatic VLDL synthesis.

Reduced fractional catabolic rate is due to decreased activity of two endothelium associated lipases namely lipoprotein lipase and hepatic triglyceride lipase which have the primary physiologic function of clearing triglycerides into FFA.

The cause of decreased lipase activities in uremia is thought to be depletion of the enzyme pool induced by

a) Heparinisation in hemodialysis[36].

b) Increase in the plasma apoc-III/apo-c II ratio[37].

c) Presence of other lipase inhibitors in the plasma.

Impaired lipase activities in uremic plasma may also be caused by a decrease in LPL synthesis as a result of secondary hyperparathyroidism or suppressed insulin level[38].

HIGH DENSITY LIPOPROTEINS:

Patients with CKD generally have reduced plasma HDL cholesterol concentration compared with nonuremic individuals. Because of the low apo-AI level and decreased LCAT activity, the esterification of free cholesterol and hence the conversion of HDL_3 to HDL_2 are diminished in uremia. This decreased ability of the HDL particles to carry cholesterol leads to impairment in the reverse cholesterol transport from peripheral cells to the liver, thereby burdening the vasculature with cholesterol and promoting atherosclerosis[39-41].

Paraoxanase, a component of HDL, inhibits the oxidation of LDL. Plasma paraoxanase activity is reduced in patients with CKD[42], thereby predisposing the LDL and possibly also

HDL particles to oxidation. Further uremia associated inflammation might convert HDL from an antioxidant into a prooxidant particle[43,44]. All of these may contribute to atherogenesis in CKD.

LOW-DENSITY LIPOPROTEIN:

Elevated plasma LDL cholesterol concentration is common in nephrotic syndromes. But it is not a typical feature of patients with advanced chronic kidney disease, especially who are on hemodialysis. But qualitative changes may occur in LDL in patients with CKD and dialysis patients.

The proportion of small dense LDL and IDL which are considered to be highly atherogenic, are increased. Sd LDL is a subtype of LDL that has high propensity to penetrate the vessel wall, becomes oxidized and triggers atherosclerotic process.

Since the hepatic lipase enzyme is decreased in HD patients, which degrades VLDL to IDL, IDL accumulates in plasma[45]. IDL and sd LDL have high affinity for macrophages, which theoretically promote their entry into the vascular wall to participate in the formation of foam cells and atherosclerotic plaques[46-49].

A vicious cycle has been suggested in uremia in which the

decreased catabolism of IDL and LDL leads to their increased plasma residence time and further modification of apo B contained in these lipoproteins by oxidation, carbamylation and glycation. These modifications lead to reduced recognition and binding of those lipoproteins to LDL receptors[50].

TOTAL CHOLESTEROL

In a prospective study[51] in 73 non diabetic patients with primary CKD,total cholesterol,LDL cholesterol and apolipoprotein B were signifigantly associated with a rapid decline in renal function.In another study[52],among 104 patients with CKD who were followed up for a mean of 4.1 years,total cholesterol and urinary protein scores were positively related to the progression of renal disease. In the Choices for Healthy Outcomes in Caring for ESRD (CHOICE) study, which showed a nonsignificant negative association of cardiovascular mortality with plasma total as well as non–HDL cholesterol levels in the presence of inflammation and/or malnutrition ; in contrast, there was a positive association between total and non–HDL cholesterol and mortality in the absence of inflammation or malnutrition. These observations are compatible with the hypothesis that the inverse association of total cholesterol levels with mortality in dialysis patients is

mediated by the cholesterol-lowering effect of malnutrition and/or systemic inflammation and not due to a protective effect of high cholesterol concentrations[53,54].But most studies state that hypercholesterolemia is not a common feature of CKD.It is commonly found in nephrotic syndrome.

LIPOPROTEIN (a)

Lipoprotein (a) is an LDL-like lipoprotein that consists of apo(a) that is covalently bound to an LDL particle. There are two isoforms of Lp(a) (i) large apo (a) isoform and (ii) small apo (a) isoform[55].

Most but not all studies showed that isoform specific increase in plasma Lp(a) levels seen in non-nephrotic patients with CKD[56-61].

Lp(a) contains 2 apolipoproteins. The production rates of apo(a) and apo(b) were normal in CKD patients whereas fractional catabolic rate of these apolipoproteins was significantly reduced in these patients. This resulted in longer residence time in plasma of almost 9 days for apo(a), compared with 4.4 days in control subjects. This decreased clearance is likely the result of loss of

kidney function in HD patients[62].

DYSLIPIDEMIA IN HEALTHY & SPECIAL POPULATION:

Dyslipidemia in Young healthy adult Indian population:

A study done by **A.M. Sawant et al**[63] in 2006 at P.D. Hinduja Hospital, Mumbai, India showed that the total cholesterol concentration ≥200mg/dl was found in 38.7% of males and 23.3% of females. HDL-C was abnormally low in 64.2% males and 33.8% females. The increase in prevalence of hypercholesterolemia and hypertriglyceridemia was more prominent in 31-40 age group than in ≤30 age group.

Dyslipidemia of Diabetes[64]

In a hospital based study in Nagpur, India, the characteristic pattern of lipoproteins in type 2 diabetes includes an increase in triglycerides and decrease in HDL cholesterol. Concentrations of LDL cholesterol in diabetic individuals do not differ significantly from concentrations found in non-diabetic individuals but are predominated by the small dense form of LDL. The small dense LDL particles are more intrinsically atherogenic than the normal larger and more buoyant LDL particles. Furthermore because of

their smaller mass, a greater number of LDL particles are contained within the plasma of patients with small dense LDL, further increase the atherogenic risk. This triad of lipid abnormalities namely increased triglycerides and sd LDL and decreased HDL, has been termed “Diabetic Dyslipidemia”.

Lipids in Haemodialysis and Peritoneal Dialysis[65-68]

HD patients usually display increased concentrations of intact or partially metabolized triglyceride rich lipoproteins, reduced serum levels of HDL cholesterol and elevated concentrations of Lp(a). Total and LDL-cholesterol values are within normal limits or reduced in this patient population whereas the sub fractionation of apoliporotein B containing lipoproteins usually reveals of predominance of small, dense LDL particles. Use of high flux polysulfone membrane is accompanied by a significant reduction in serum triglyceride levels as well as by an increase in apolipoprotein AI, and HDL cholesterol levels . This improvement could be attributed to an increase in the apolipoprotein C-II/CIII ratio which increases the activity of lipoprotein lipase and facilitates the intravascular lipolysis of triglyceride rich lipoproteins . Heparin, used in HD, releases lipoprotein lipase from the endothelial surface

and thus its chronic use may result in lipoprotein lipase depletion and defective catabolism of triglyceride rich lipoproteins.

It is well known that CAPD patients lose substantial amount of proteins into peritoneal dialysate. This protein loss may, in turn, stimulate hepatic production of albumin and cholesterol enriched lipoproteins thus leading to elevated concentrations of LDL cholesterol and Lp(a) . In addition, the absorption of glucose from the dialysis fluid and the resultant increase in insulin levels may enhance the hepatic synthesis and secretion of VLDL and possibly that of other lipoproteins such as Lp(a) .

Lipids in renal transplant patients[69]

These patients have elevated values of total cholesterol, VLDL, LDL cholesterol as well as increased concentration of triglycerides and apolipoprotein B . HDL cholesterol tends to increase in post transplant period and this change is attributed to the effects of corticosteroids. They also exhibit significant decrease in the concentrations of Lp(a) after renal transplantation. It has been shown that cyclosporine administration significantly increases the concentrations of LDL-C and TGL while it reduces the serum

values of HDL cholesterol .

Treatment of dyslipidemia in CKD patients[69]

Data from studies conducted in individuals with CKD suggest that the effect of these drugs on cardiovascular morbidity and mortality in these patients is significantly influenced by the severity of renal dysfunction. Thus in several large, prospective, placebo controlled trials of statins, post hoc analyses of subgroups with mild to moderate renal failure revealed a significant reduction in cardiovascular morbidity and mortality . The use of statins as a first line therapy for the prevention of ischemic events in dyslipidemic individuals with CKD (stage 1-3) seems to be safe, reasonable and evidence based.

It has been proposed that the failure of statins in ESRD patients is due to the presence of micro inflammation and malnutrition in these individuals .

Though fibrates induce shift in the LDL subfraction towards larger and more buoyant particles , in patients with renal failure they are associated with high risk of muscular toxicity . Fibrates should be used only in the patients with CKD who exhibit

extremely elevated triglyceride values (>500mg/dl). In these cases the risk of acute pancreatitis justifies the use of gemfibrozil as the fibrate of choice in individuals with impaired renal function .

Efficiency of other drugs in patients with CKD and their impact on the cardiovascular risk in these patients were not studied well.

MATERIALS & METHODS

This study was conducted in 50 patients with chronic kidney disease and 50 normal healthy persons.

All the patients in this study group were selected from the outpatient department and those who were admitted to **Institute of Internal Medicine, Madras Medical College Hospital** during June2007 - June 2008. The controls were selected from the outpatient department who were accompanying the patients.

STUDY DESIGN: Cross sectional observational study

INCLUSION CRITERIA FOR PATIENTS

1. Patients between age group of 15 to 80 years with chronic kidney disease.
2. Patients with established chronic kidney disease were selected irrespective of the etiology.
3. Patients who were on conservative or dialytic treatment for chronic kidney disease.
4. Established renal failure was ensured by radiological evidence or biochemical evidence for more than 3 months.

EXCLUSION CRITERIA

1. Patients with Acute renal failure and Nephrotic Syndrome

2. Who are on drugs affecting lipid metabolism like β blockers, statins and oral contraceptive pills.

3. Female patients who were pregnant

Written consent was obtained from both patients and controls.

Detailed history regarding symptoms and duration of the kidney disease, hypertension, diabetes, smoking, alcoholism, drug intake and treatment were elicited. A detailed clinical examination was performed in all patients. Blood pressure, renal function tests, abdominal ultra sonogram and Electrocardiogram were done for all patients.

After 12 hours of overnight fasting blood sample was taken for lipid profile from patients and controls.

Patients with chronic kidney disease and controls included in the study were matched according to age and the results were analyzed.

LABORATORY METHODS FOR ESTIMATING LIPIDS

HDL-C Estimation: (Erba Mannheim - XL System packs)

HDL cholesterol was measured by using immunoinhibition method. The reagent contains 2 parts. First reagent (R1) inhibits lipoprotein fractions other than HDL-C. After adding reagent 2(R2), a blue colour complex develops. The intensity of blue color complex formed at 593nm is proportional to the HDL-C in the sample.

Principle

LDL, VLDL & Chylomicrons in the sample were complexed with antibody present in the reagent 1 thereby HDL-C remains free to react with reagent 2.

$$\text{HDL cholesterol} + H_2O + O_2 \xrightarrow[\text{in reagent 2}]{\text{CHE \& CO}} \text{4-Cholestenone} + \text{Fatty Acid} + H_2O_2$$

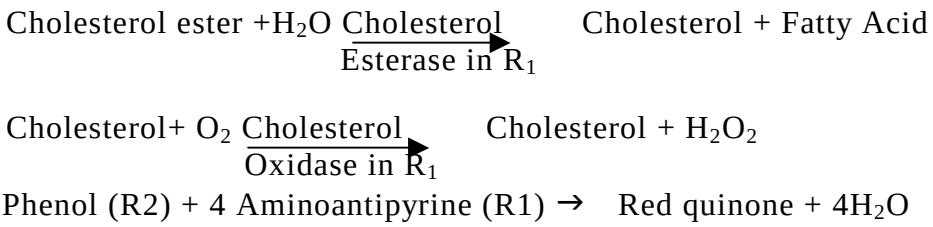
$$H_2O_2 + \text{DAOS} + \text{4 AAP} \xrightarrow{\text{Peroxidase}} \text{Blue colored complex} + 2H_2O$$

Cholesterol estimation (Autopack - Bayer)

Cholesterol was estimated by enzymatic method. The reagent contains 2 parts. (R_1 & R_2). When R1 is added it forms hydrogen peroxide. This in turn reacts with phenol in R_2 and forms red quinone. The intensity of red coloured complex is directly

proportional to the concentration of cholesterol in the sample. It is measured at 500nm.

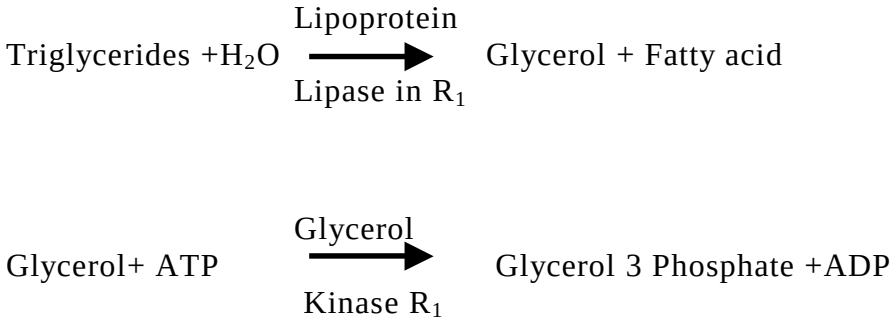
Principle:

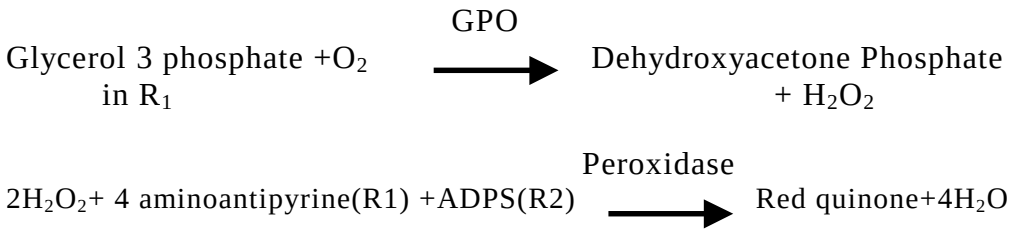


TGL Estimation (Bayer Diagnostics)

It was measured by enzymatic calorimetric method. The reagent contains 2 parts. When R_1 is added to the sample, H_2O_2 is formed. This in turn reacts with R_2 and forms red quinone. The intensity of purple colour is directly proportional to the triglyceride concentration in the sample and is measured at 546nm.

Principle:





ADPS = N ethyl N sulfopropyl n-anisidine.

LDL was estimated by using **Friedwald** formula

LDL = Total cholesterol - (HDL-C + TGL/5)

Ultra sonogram showing reduced kidney size (<9cm) was taken as radiological evidence of chronic kidney disease.

Electrocardiogram of all patients was studied in detail.

Romhilt- Estes criteria was applied for finding out left ventricular hypertrophy.

ROMHILT ESTES SCORING SYSTEM FOR LVH

		Points
1.	R or S wave in any limb lead ≥ 2mv	3
	or S in lead V1 or V2	
	or R in lead V5 or V6 ≥ 3mv	
2.	Left Ventricular strain	
	ST segment and T wave in opposite direction to QRS complex	
	Without digitalis	3
	With digitalis	1
3.	Left atrial enlargement	
	Terminal negativity of the P wave	3
	in lead V1 is ≥ 0.10 mV in depth	
	and ≥ 0.04 S in duration	
4.	Left axis deviation ≥ -30 degrees	1
5.	QRS duration ≥ 0.09 seconds	1
6.	Intrinsicoid deflection in lead	
	V5 or V6 ≥ 0.05 S	1
	Maximally attainable	13

LVH - 5 points

Probable LVH-4 points

ATP - III NCEP guidelines were applied to the lipid profile.

ATP III CLASSIFICATION OF LDL, TOTAL AND HDL CHOLESTEROL AND TRIGLYCERIDES

TOTAL CHOLESTEROL	
<200	Desirable
200-239	Borderline high
>240	High

LDL CHOLESTEROL	
<100	OPTIONAL
100-129	Near or above normal
130-159	Border line High
>160	High

HDL CHOLESTEROL	
<40	Low
>60	High

TRIGLYCERIDES	
<150	Normal
150-199	Borderline high
200-499	High
≥500	Very high

In this study

LDL > 130 mg/dl

HDL < 40 mg/dl

TGL > 200 mg/dl

TC > 240 mg/dl were considered abnormal

STATISTICAL METHODS

Mean values were obtained for LDL, HDL, TGL & Total cholesterol separately. Then standard deviations were calculated for each category of observations for both study and control group. Mean deviation, standard error of difference between two means was calculated. If the standard error of difference between two means is more than two times that of actual difference between two means it will be taken as significant value. Students T test was performed & T value was obtained. P value from t value was calculated. P value of <0.05 was considered significant

RESULTS AND OBSERVATIONS

AGE DISTRIBUTION

Age of the patients varied from 14 yrs to 80 yrs. Majority of patients fall in the age group between 26-55 years. Seventy two percentage of people contribute this group.

AGE DISTRIBUTION IN PATIENTS

Age in years	*No. of Patients*	*Percentage*
15-25	2	4%
26-35	13	26%
36-45	11	22%
46-55	12	24%
56-65	10	20%
66-75	1	2%
>75	1	2%

SEX DISTRIBUTION

Males constitute 34(68%) and females constitute 16(32%) in this study.

EDUCATIONAL STATUS

Majority of patients were illiterate (23 persons) (46%). Patients studied higher than HSc were 6 persons (12%). Others studied between 5^{th} std to 10^{th} std.

OCCUPATION

Patients in this study belonged to low socioeconomical status which is being reflected by their occupations. A quarter of the patients were doing agriculture or agriculture related profession. Most women were home makers.

PERSONAL HABITS

In this study 40% (20 patients) of the patients were smokers and 42 % (21 patients) were alcoholics.

RENAL PARAMETERS

Lowest urea value found in these patients was 17mg/dl and the highest was 230 mg/dl. Creatinine values ranged between 0.5 mg/dl to 22.6 mg/dl. Mean values of creatinine was 6.74 ± 4.77 mg/dl.

BLOOD PRESSURE READINGS

Patients with blood pressure of more than 140/90 were considered hypertensives. Most patients (39 patients) were hypertensives at the time of presentation. Only 11 patients had blood pressure less than 140/90mmHg.

DIABETIC STATUS

It was found that 22 patients (44%) were diabetic and their random blood sugar levels ranged from 51mg/dl to 388 mg/dl.

TREATMENT SCENARIO

Among the 50 patients, 33 patients (66%) were on conservative treatment only, 10 patients (20%) received peritoneal dialysis, 7 patients (14%) received hemodialysis. Out of the 10 patients who underwent PD, 7 patients underwent PD earlier than 1 month ago and only 3 patients were treated with PD during the time of admission. In the HD group, 4 patients underwent dialytic treatment a week before admission whereas other 3 received more than a month ago. 66% patients were treated with drugs only. During our study no patients were admitted with history of previous transplantation.

RADIOLOGICAL EXAMINATION

Radiological examination was done by abdominal ultra sonogram. In 38 patients the kidney size was less than 9cm in one or both kidneys. Rest of them (12 patients) showed normal kidney size in USG.

ECG RESULTS

Patient's electrocardiograms were analysed. It was found that 15 patients (30%) showed left ventricular hypertrophy. 10 patients (30%) showed ischemic changes. 3 patients showed tall peaked T waves.

ECG CHANGES IN CKD PATIENTS (STUDY GROUP)

Type of ECG changes	***Males***	***Females***	***Combined***
LVH	10(20%)	5(10%)	15(30%)
Ischemia	6(12%)	4(8%)	10(20%)

CKD PATIENTS WITH LVH SHOWING LIPID ABNORMALITIES (STUDY GROUP)

Type of lipid Disorders	***Number of Patients (out of 15)***	***Percentage***
Elevated Cholesterol	3	20%
Elevated Triglycerides	4	27%
Decreased HDL	10	67%
Increased LDL Cholesterol	10	67%

CKD PATIENTS WITH ISCHEMIA SHOWED THE FOLLOWING LIPID DISORDER

Type of lipid Disorders	*Number of Patients (our of 10)*	*Percentage*
Elevated Cholesterol	3	30%
Elevated Triglycerides	7	70%
Decreased HDL	6	60%
Increased LDL Cholesterol	6	60%

LIPID PATTERN IN OUR STUDY

HDL PATTERN

Serum HDL values ranged between 30mg/dl to 80mg/dl. Patients showed abnormal HDL levels (<40 mg/dl) were 25 (50%). Its mean value was 42.82 and standard deviation was 12.25. Among the control groups, the lowest value of HDL was 46 mg/dl and the highest was 65 mg/dl. Their mean was 54.20 and standard deviation was 4.18. Mean deviation and standard error of difference between two means were calculated. Actual difference between two mean was 8.07 and the standard error of difference between two means was 1.83. This was statistically significant since the actual difference was two times higher than the standard error of

difference between two means. T value was calculated using student's t test. It was 6.2169. P value (<0.05) was statistically significant. It showed that there was a significant reduction in HDL-C levels in patients with CKD than that of controls.

MEAN AND STANDARD DEVIATION OF LIPID FRACTIONS IN 50 CKD PATIENTS IN THE STUDY

	Total Cholesterol mg/dl	***Triglycerides mg/dl***	***Low Density Lipoprotein mg/dl***	***High Density Lipoprotein mg/dl***
Mean	209.3	171.2	131.7	42.82
Standard Deviation	42.9	86.45	25.71	12.25

MEAN AND STANDARD DEVIATION OF LIPID FRACTIONS IN 50 CONTROLS

	Total Cholesterol mg/dl	***Triglycerides mg/dl***	***Low Density Lipoprotein mg/dl***	***High Density Lipoprotein mg/dl***
Mean	185.2	102.2	112.5	54.20
Standard Deviation	15.2	7.75	13.42	4.18

STANDARD ERROR OF DIFFERENCE BETWEEN TWO MEANS AND P VALUES

	Total Cholesterol mg/dl	***Triglycerides mg/dl***	***Low Density Lipoprotein mg/dl***	***High Density Lipoprotein mg/dl***
Standard error of Difference between two means	6.44	12.28	3.61	1.83
P values	<0.05	<0.05	<0.05	<0.05

LDL PATTERN

Lowest value of LDL 65 mg/dl and the highest value was 173mg/dl. Abnormally high LDL levels (>130mg/dl) were found in 22 patients and they constitute 44%. Their mean value was 131.7 mg/dl and standard deviation was 25.71.In controls, the mean and SD were 112.5 and 13.42 (Range 85 - 150mg) respectively.

Standard error of difference between two means was 3.61. Actual difference between two means was 19.2 which were (20%) two times greater than the standard error of difference between two means. Student t value was calculated (t=4.6813) and P value was (< 0.05) significant.

TGL PATTERN

TGL value in our study group ranged between 95 mg/dl to 350 mg/dl. Range of TGL value in control group was 90mg/dl to 122mg/dl. TGL levels were abnormal in 24 patients (>200mg/dl). Mean and standard deviation of study group were 171.2 and 86.45 respectively. In controls, the mean and standard deviation were 102.2 and 7.75. Student‘t’ test was performed and t value was calculated (t=5.6212). P value was significant ($P < 0.05$)

TOTAL CHOLESTEROL

Range of TC levels in study group was 120mg/dl to 258 mg/dl. Lowest value in control group was 119 and the highest value was 222mg/dl. Total cholesterol was more than 240mg/dl in 10 patients (20%). The mean values of study group and control group were 209.3 and 185.2mg/dl respectively. Their standard deviations were 42.9 and 15.2 respectively. Standard error of difference between two means was obtained. It was 6.44 but the actual difference was 124.1 which was more than two times higher than that of standard error of difference between the two means. T value was calculated (t =3.7442). P value was ($P<0.05$) significant.

CORRELATION STUDIES

CORRELATION BETWEEN LIPID FRACTIONS AND SERUM CREATININE IN PATIENTS

Lipid Fraction	Correlation coefficient with Creatinine	P Value
TC	+0.12469	N.S
TGL	+0.23731	<0.05
HDL	-0.28328	N.S
LDL	+0.21606	N.S

N.S - Not Significant

Correlation coefficient between serum creatinine and various lipid fractions were calculated.

Correlation coefficient between serum creatinine and LDL was +0.216 which was a positive correlation.

Correlation coefficient between HDL and creatinine was – 0.286, which was a negative correlation.

Correlation coefficient between creatinine and TG was +0.124 which was a positive correlation.

Correlation coefficient between creatinine and total cholesterol was +0.12469, which was a positive correlation.

P values from the correlation coefficient were calculated.

P value of correlation coefficient between serum creatinine and HDL levels was statistically significant in the study group. It indicates that there is a negative linear relationship exists between HDL values and serum creatinine (i.e. when creatinine value raises HDL value falls).

DISCUSSION

In our study, most common lipid abnormalities found were low HDL levels (50%) and hypertriglyceridemia (48%).

COMPARISON OF LIPID PROFILE BETWEEN OTHER STUDIES AND OUR STUDY

Studies		*TGL*	*LDL*	*HDL*	*TC*
Shah et al	S	222.78±90.08	109.63±36.51	52.69±16.36	211.33±40.33
	C	121.78±64.89	140.33±23.34	44.22±10.33	184.11±18.79
Diana M lee et al	S	194.05±106.28	170.148±50.27	38.6±11.6	239.75±61.8
	C	106.28±26.5	131.47±30.93	42.53±7.73	189.14±30.93
Our study	S	171.2±86.45	131.7±25.71	42.82±12.25	209.3±42.9
	C	102±7.5	112.5±13.42	54.20±4.18	185.2±15.2

S- Study Group C-Control Group

DECREASED HIGH DENSITY LIPOPROTEIN LEVELS

The low HDL levels in patients with chronic kidney disease in our study were consistent with **Diana M Lee LG et al**[70] who studied the lipid profile in CRF patients.

This low HDL cholesterol levels were also an independent risk factor for the development of CKD in the Framingham off spring study.

Several mechanisms may underlie these reductions in HDL cholesterol levels, which is usually an indication of impaired reverse cholesterol transport. Thus, uremic patients usually exhibit decreased levels of apolipoprotein AI & AII (the main protein constituent of HDL). Diminished activity of LCAT (the enzyme responsible for the esterification of free cholesterol in HDL particles) as well as increased activity of cholesterol ester transfer protein that facilitates the transfer of cholesterol esters from HDL to TGL rich lipoproteins that reduce serum concentrations of HDL cholesterol[69].

In MDRD study[71], low HDL levels in CKD patients were one of the independent risk factor for progression of kidney disease. Though in our study the mean value was 42.82, it is significantly less than the age matched healthy controls.

VARIOUS STUDIES ON PROGRESSION OF KIDNEY DISEASE AND ASSOCIATED PLASMA LIPID ABNORMALITIES:

Study	*Patients*	*Number of patients*	*Follow up*	*Lipid*
MDRD	CKD	840	2.2 YRS	↓HDL
Samuelsson O et al	CKD	73	3.2 YRS	↑TCh, ↑LDL, ↑ApoB
Locatelli et al	CKD	456	2 YRS	No relationship
Massy ZA et al	CKD	138	12 YRS	↑TG, ↓HDL

ELEVATED TRIGLYCERIDES

Hypertriglyceridemia was observed in 48% of patients. Triglyceride levels were significantly elevated in our study than control group. Abnormal triglyceride values were found in 48% of patients in our study. **Shah et al**[72] & most western studies demonstrated that hypertriglyceridemia was the abnormality found in CKD patients. **Gupta DK et** al[6]**, Das BS et** al[7]**, Bagdade J**[73]**,**

Chan MK et al[74] also found hypertriglyceridemia was the major abnormality in their studies.

Hypertriglyceridemia represents an early feature of renal failure. Indeed previous studies have shown that patients with impaired renal function exhibit increased concentrations of triglycerides even though serum creatinine levels were within normal limits.

In addition, individuals with renal insufficiency, usually display abnormal increase in serum triglycerides levels after a fat meal (post prandial lipemia). Experimental studies revealed that accumulation of triglyceride rich lipoprotein (VLDL, chylomicrons and their remnants) in individuals with predialysis CKD is mainly due to their decreased catabolism. The down regulation of the expression of several genes along with the changes in the composition of lipoprotein particles and the direct inhibitory effect of various uremic toxins on the enzymes involved in lipid metabolism represent the most important pathophysiological mechanism underlying the development of hypertriglyceridemia in renal failure. Interestingly, it has been proposed that secondary hyperparathyroidism may also contribute to the impaired

catabolism of triglyceride rich lipoproteins and that parathyroidectomy or the administration of calcium channel blocker verapamil may partially ameliorate the hypertriglyceridemia of CKD. It is well known that impaired insulin sensitivity represents an early feature of CKD. Thus it could be due to insulin resistance driven over production of VLDL may significantly contribute to the development of hypertriglyceridemia in CKD patients[70].

ELEVATED LOW DENSITY LIPOPROTEIN

LDL was significantly elevated than that of controls in our study. We found that 44% of patients showed elevated LDL levels. This observation is similar to the studies of **Diana M Lee et al**. In an article published in archives of internal medicine[75] 32 patients were studied and compared the lipid profile on CKD and non CKD patients. It was found that 60.5% of patients have elevated LDL-C than non CKD patients (P=0.06). But most studies find that Uremic Patients usually have normal or slightly reduced concentrations of LDL-C levels and they exhibit important disturbance in the density distribution of LDL sub fraction that is characterized by a predominance of small dense LDL particles[76-80].

In our study, this elevated LDL-C may be due to the inaccuracy of Friedwald formula in estimating LDL cholesterol. Another contributing factor is that peritoneal dialysis may increase LDL-C due to the compensatory production of lipoprotein in response to protein loss during the procedure.

Total Cholesterol:

Total cholesterol levels were significantly elevated in our study group. We observed the same findings in the study by **Diana Lee M et al**. But most of the studies did not observe hypercholesterolemia. The possible reason for the hypercholesterolemia in our study is significant elevation of cholesterol containing lipid fractions (IDL, LDL).

Correlation Studies:

It was found that serum triglycerides, TC, LDL, were not correlated significantly whereas serum HDL levels had significant negative correlation with serum creatinine. It means that when serum creatinine level rises, serum HDL level falls. This was the observation found in MDRD study.

ECG changes:

Out of 50 patients, 30% of patients showed changes suggestive of LVH and 20% of patients showed ischemic changes. This observation was similar to study done by **Levin et al**. The risk of dying of cardiac complications is 65 times higher in dialysis patients between 45-54 years and 500 times higher than the general population. The risk factors which are responsible for increased morbidity and mortality were hypertension, DM, high LDL, low HDL, smoking, LVH, male gender, old age, anemia, hypervolemia, insulin resistance and proteinuria.

CONCLUSION

1. HDL-C levels were lower and triglycerides, total cholesterol and LDL-C levels were higher in the study group compared to controls. All were statistically significant.

2. Predominant lipid abnormality was reduced HDL-C levels.

3. There was a negative correlation exists between serum HDL-C level and serum creatinine levels which was statistically significant.

4. Percentage of patients showing ECG changes of left ventricular hypertrophy and ischemia were 30% and 20% respectively.

LIMITATIONS OF THE STUDY

1. Most studies showed low or normal LDL-C levels whereas this study observed significantly elevated LDL-C levels for reasons little known.

2. Smoking, alcoholism and diabetics may alter the lipid pattern in the body. Their influences in the study group also have to be considered.

3. Since we had not analysed the echocardiogram of the patients, the real scenario of ischemia in CKD patients who had not shown any abnormality in ECG was not known.

4. We had not estimated the lipid abnormalities in patients who underwent dialytic treatment since most of them were irregular in their treatment.

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LIPID PROFILE IN PATIENTS WITH CHRONIC KIDNEY DISEASE

Sl. No	*Name*	*Age*	*Sex*	*HDL*	*LDL*	*TGL*	*TC*
1.	Narayanan	44	M	80	126	180	242
2.	Guthigan	44	M	42	150	230	238
3.	Sundaramoorthy	30	M	55	120	146	206
4.	Kumar	27	M	32	162	220	238
5.	Subrammani	65	M	50	128	214	220
6.	Babu Sethu	32	M	32	126	96	177.2
7.	Shankar	30	M	68	146	220	258
8.	Chokkammal	45	F	35	119	110	176
9.	Amaravathy	65	F	35	65	104	120.8
10.	Suseela	30	F	30	140	350	240
11.	Narayana Samy	55	M	45	168	160	245
12.	Mariyan Beevi	60	F	76	130	185	243
13.	Kumar	29	M	32	156.8	106	210
14.	Banu	27	F	48	128	150	206
15.	Chinna Ponnu	45	F	45	151	220	240
16.	Chinna Samy	55	M	40	148	235	235
17.	Saratha	37	F	48	130	200	218
18.	Ponmudi	52	M	35	65	104	120
19.	Sithiraj	55	M	32	85	95	136
20.	Ibrahim	38	M	42	86	110	152
21.	Vasantha Kumar	24	M	36	150	240	234
22.	Marimuthu	50	M	42	147	200	229
23.	Kondai raj	32	M	76	125	190	239
24.	Panja charam	64	M	40	126	130	202
25.	Selvi	40	F	40	123	210	205

Sl. No	*Name*	*Age*	*Sex*	*HDL*	*LDL*	*TGL*	*TC*
26.	Ramu	60	M	36	131	235	214
27.	Saratha	35	F	38	158	260	248
28.	Ramachandran	80	M	40	167	158	238
29.	Kalyanam	40	M	36	112	100	168
30.	Lurthu samy	50	M	36	110	110	168
31.	Orammal	55	F	40	149	206	230
32.	Siva Kumar	38	M	36	158	220	238
33.	Geetha	45	F	36	160	228	241.6
34.	Jeveula	70	M	36	112	100	168
35.	Mani	52	M	40	122	209	222
36.	Senthil	29	M	36	135	95	190
37.	Govindaraj	55	M	40	167.8	136	235
38.	Arumugam	60	M	73	125	218	242
39.	Vasantha	35	F	40	126	200	202
40.	Ellappan	57	M	35	119	110	176
41.	Perumal	70	M	39	148	200	227
42.	Vijayalakshmi	47	F	38	173	95	230
43.	Kannagi	40	F	36	112	100	168
44.	Ramesh	32	M	38	149	205	228
45.	Rajathi	38	F	36	110	110	168
46.	Mathivanan	50	M	38	170	136	235.5
47.	Kalaiyarasan	35	M	66	148	205	255
48.	Ayyaru	65	M	40	128	208	209.6
49.	Pichai Kannu	60	M	36	66	104	122.8
50.	Anjammal	56	F	40	128	206	210

LIPID PROFILE IN CONTROLS

S.No	*Name*	*Age/Sex*	*TGL mg%*	*LDL mg%*	*HDL mg%*	*TL mg%*
1.	Anbuselvam	35/M	106	108	58	186.8
2.	Ponnammal	47/F	95	120	55	194
3.	Kumar	37/M	90	128	50	196
4.	Parimala	40/F	105	114	60	195
5.	Karitha	22/F	110	150	50	222
6.	Raju	60/M	95	125	55	119
7.	Chinnakannu	72/M	90	128	58	204
8.	Gangadharan	48/M	115	106	65	194
9.	Deva Anbu	37/M	106	107	50	178.2
10.	Sironmani	42/M	102	103	53	176.4
11.	Kannaiya	54/M	115	110	54	176.4
12.	Subramaniyan	38/M	90	120	50	187
13.	Rajeshkumar	25/M	105	110	60	191
14.	Maniyammal	39/F	102	124	56	200.4
15.	Bharathi Devi	28/M	100	115	54	189
16.	Neela	47/F	100	85	55	160
17.	Panneerselvam	50/M	102	103	55	178
18.	Velmurugan	33/M	96	128	50	192
19.	Poongothai	37/F	100	125	55	200
20.	Savithiri	35/F	116	102	55	180
21.	Sadasivam	37/M	90	120	50	180
22.	Perumal	47/M	112	100	50	188
23.	Selvi	30/F	96	128	51 (50)	192

24.	Nalini	31/F	95	116	58	193
25.	Murugesan	62/M	100	125	55	201
26.	Pavithra	19/F	116	102	55	180
27.	Narmatha	33/F	102	104	56	180.4
28.	Kaliyaperumal	75/M	90	120	50	188
29.	Pitchaikannu	47/M	94	120	55	193.8
30.	David	39/M	96	104	55	178.2
31.	Gunasekaran	34/M	98	103	55	177.6
32.	Harini	27/F	100	128	46	194
33.	Veerasekar	67/M	104	98	60	178.8
34.	Vikram	38/M	103	98	60	178.8
35.	Elangovan	35/M	98	98	56	173.6
36.	Pathiyammal	78/F	109	100	58	179.8
37.	Porselvi	40/F	110	114	60	196
38.	Kanagavalli	26/F	98	100	54	1736
39.	Muthukumar	24/M	100	106	49	175
40.	Stalin	30/M	104	90	48	158.8
41.	Sampathkumar	37/M	102	108	58	186.4
42.	Nagarani	55/F	105	90	56	167.0
43.	Anjammal	58/F	110	92	58	172
44.	Valli	43/F	100	106	55	180
45.	Vadivammal	58/F	106	105	55	182
46.	Ravi	38/M	122	124	50	198
47.	Mathialagan	50/M	118	107	50	180
48.	Govindaraju	56/M	100	128	48	196
49.	Kalaivanan	29/M	95	143	48	210
50.	Vani	32/M	96	125	50	194.2

PROFORMA

No.:

Name: Age/Sex I.P. No. DOA Date

Unit: Education: Occupation:

RFT

Sugar Na+ TC PCV

Urea K+ DC P L E Hb

Creatinine ESR Platelets

USG Abdomen: RK PCS

LK PCS Pancreas

Other Findings

BP Status Glycemic Status

Drugs

Duration of DM - Drugs/Insulin/Nil

Duration of HTN - Drugs/No Drugs/Irregular

Other causes of CKD

H/o Smoking Symptoms Signs

H/o Alcoholism

Family H/o Kidney Disease: DM / HTN

Daily urine volume now:

On drugs only / PD / HD / Transplant - If dialysis no. of times last dialysis

Date of Transplant

ECG ECHO if done

Lipids: TGL TC HDL LDL

MASTER CHART

Sl. No	*Name*	*Age*	*Sex*	*IP. No*	*Education*	*Occupation*	*Smoking*	*Alcohol*	*Na+meq/L*	*K+ meq/L*	*Urea mg%*	*Creatinine mg%*	*Sugar mg%*	*BP mmhg*	*DM*	*HTN*	*Treatment /last dialysis*	*HDLmg/dl*	*LDL mg/dl*	*TGL mg/dl*	*TC mg/dl*	*RK size (cm)*	*LK size (cm)*	*PCS*	*ECG Changes*
1.	Narayanan	44	M	65815	8th Std	Leather Works	+	-	11.5	3.6	48	1.8	60	150/100	–	+		80	126	180	242	10	11	N	N
2.	Guthigan	52	M	65178	–	Agri	+	+	120	5	44	1.8	66	150/100	–	–	Drugs	42	150	230	238	10	9.5	N	N
3.	Sundaramoorthy	30	M	70240	–	Wood cutter	–	–	138	3.8	55	3.6	55	120/70	–	–	HD/ 5 days ago	55	120	146	206	8.1	8.1	Dilated/ B/L	N
4.	Kumar	27	M	70208	–	Painter	–	+	118	4.2	230	14.5	110	160/100	–	+	PD / 2 months	32	162	220	238	8.5	7.2	N	T↓ V1-V4
5.	Subrammani	65	M	67166	2nd std	Agri	+	+	130	4	70	2.8	136	130/100	+	+	PD / 1 month	50	128	214	220	7.5	8.8	N	LVH with I, avl; ST↓ V5-V6
6.	Babu Sethu	32	M	65742	SSLC	Flower Business	–	+	126	5	192	22.6	80	160/100	–	+	Drugs	32	126	96	177.2	7.5	7.3	N	LVH with strain
7.	Shankar	30	M	67047	SSLC	Driver	–	–	134	4	135	12.3	95	180/110	+	+	HD/ 4 days	68	146	220	258	8.5	8.8	N	LVH
8.	Chokkammal	45	F	66802	–	Home maker	–	–	118	6	47	1.5	51	90/60	+	–	Drugs	35	119	110	176	10.2	8.9	N	Sinus tachy cardia

9.	Amaravathy	65	F	66803	–	Home maker	–	–	127	4.8	64	2.3	192	160/100	+	+	Drugs	35	65	104	120.8	8.5	9.8	N	N
10.	Suseela	30	F	66312	–	Home maker	–	–	Low	4.5	151	13.9	57	160/100	–	–	PD/ 1 month	30	140	350	240	7.3	7.1	N	N
11.	Narayana Samy	55	M	68068	–	Agri	–	–	118	5.6	192	12	94	180/100	+	–	PD/ 2 months	45	168	160	245	6.2	6.3	N	LVH with ST↓ II, III, AVF
12.	Mariyan Beevi	60	F	67653	–	Home maker	–	–	126	4.9	68	2.5	104	190/100	+	+	Drugs	76	130	185	243	10.3	11.7	N	LAD/ LVH
13.	Kumar	29	M	67957	8th std	Business	+	+	132	4.2	221	14.3	100	240/140	–	–	Drugs	32	156.8	1.6	210	7.6	8	N	LVH
14.	Banu	27	F	68271	7th std	Agri	–	–	Low	5.9	174	11.3	135	150/80	–	+	Drugs	48	128	150	206	8.2	8.3	N	LVH / T↓ V4-V6
15.	Chinna Ponnu	45	F	68307	–	Agri	–	–	133	5.8	76	3.6	118	140/90	+	–	Drugs	45	151	220	240	7.1	7.6	N	N
16.	Chinna Samy	55	M	71885	SSLC	Agri	–	–	Low	5	148	2.9	102	140/100	–	+	PD/ 3 months	40	148	235	235	7.2	7.5	N	T↓ I, AVL; QS V1-V6
17.	Saratha	37	F	69638	5th std	Home Maker	–	–	130	4.8	116	5	96	160/90	–	–	PD/ 2 months	48	130	200	218	7.8	7.2	N	N
18.	Ponmudi	52	M	72136	–	Construction worker	+	+	110	3.2	120	4.8	82	150/100	–	+	HD 1 day	35	65	104	120	8.4	83.6	N	N
19.	Sithiraj	55	M	68411	–	Agri	+	+	128	4	79	2.8	92	130/90	–	+	Drugs	32	85	95	136	9.4	9	N	Low voltage complex
20.	Ibrahim	38	M	66059	8th std	Leathar worker	–	–	115	3.5	140	8	154	130/60	–	–	Drugs	42	86	110	152	12	12	N	N

21.	Vasantha Kumar	24	M	66105	B com	BPO	–	–	128	6.4	185	21.2	200	200/110	–	–	Drugs	36	150	240	234	6.9	7.2	N	N
22.	Marimuthu	50	M	70221	12th std	Agri	+	+	118	5.3	123	7.4	129	140/100	+	–	HD/1 month	42	147	200	229	11.4	11.3	N	Tall T Waves
23.	Kondai raj	32	M	65293	4th std	Load man	+	+	124	3.8	17	0.5	100	190/120	–	–	HD/ regular	76	125	190	239	8.3	7.8	N	LVH
24.	Panja charam	64	M	65007	8th std	Lorry driver	+	+	131	5.7	102	5.6	104	130/80	–	+	Drugs	40	126	130	202	7	8.1	N	Sinus Brady cardia
25.	Selvi	40	F	66057	–	Home maker	–	–	124	5.1	68	5.6	388	130/100	+	+	Drugs	40	123	210	205	8.4	7.2	N	ST↓ I; AVL, V5-V6
26.	Ramu	60	M	66032	5th std	Washer man	–	–	118	6.2	177	14.6	122	200/120	–	–	Drugs	36	131	235	214	7.8	7.4	N	Tall T waves
27.	Saratha	35	F	64541	–	Home maker	–	–	131	4	176	10.2	97	200/120	+	–	PD/ 5 days	38	158	260	248	8.1	9.6	N	Poor progression R wave, V1-V3
28.	Ramachandran	80	M	70055	3rd std	Agri	+	+	124	3	112	5.6	80	140/90	+	+	Drugs	40	167	158	238	6.8	7.2	N	LVH
29.	Kalyanam	40	M	70118	7th std	Agri	+	–	128	5	76	3.4	132	221/50	+	–	Drugs	36	112	100	168	9.4	9.8	N	N
30.	Lurthu samy	50	M	69928	6th std	Mason	–	–	128	3.2	143	7.6	72	150/90	+	+	Drugs	36	110	110	168	6.1	6.3	N	N
31.	Orammal	55	F	65139	9th std	Home maker	–	–	116	5.5	120	6	120	140/90	+	+	Drugs	40	149	206	230	10.1	10.8	N	LVH
32.	Siva Kumar	38	M	68315	12th	Agri	+	+	130	5	148	7	110	150/100	–	+	Drugs	36	158	220	238	7.1	7.8	N	N

					std																			
33.	Geetha	45	F	67412	PUC	Home maker	–	–	116	3.8	110	5.8	100	130/100	–	–	Drugs	36	160	228	241.6	8.2	7.8	N	N
34.	Jeveula	17	M	72163	10th std	Carpenter	–	–	118	5	100	5.5	106	130/80	–	+	Drugs	36	112	100	168	6.8	7.1	N	LVH
35.	Mani	52	M	66034	–	Business	+	+	126	5	140	7	150	200/100	+	+	HD/ 2 months	40	122	209	222	9.8	10.4	N	N
36.	Senthil	29	M	70106	BSc	Clerk	–	+	Low	6	124	7	128	140/100	+	+	Drugs	36	135	95	190	7.8	11.2	N	LVH
37.	Govindaraj	55	M	67875	7th std	Steel work	+	–	130	5.6	112	5.5	80	200/120	+	–	Drugs	40	167.8	136	235	6.4	7.2	N	T ↓ V4-V6
38.	Arumugam	60	M	68130	5th std	Painter	+	+	127	4.8	120	6	110	140/90	+	+	PD/3 days	73	120	218	242	9	10.2	N	ST ↓ V1-V4
39.	Vasantha	35	F	65235	SSLC	Home Maker	–	–	130	7	140	6.7	78	190/120	–	+	Drugs	40	126	200	202	8.4	8.6	N	Tall T waves
40.	Ellappan	57	M	67315	–	Agri	+	+	116	4.5	148	7	112	150/100	–	+	PD/1 month	35	119	110	176	6.9	7.4	N	N
41.	Perumal	70	M	68112	–	Mason	+	+	130	5	112	6	100	130/90	–	+	Drugs	39	148	200	227	7.1	7.8	N	N
42.	Vijayalakshmi	47	F	68306	–	Construction worker	–	–	134	4	70	3	90	150/90	–	+	Drugs	38	173	95	230	7.4	9	N	PRWP V1-V4
43.	Kannagi	40	F	66720	7th std	Agri	–	–	116	3.9	123	5.4	116	130/90	–	–	Drugs	36	112	100	168	8	8.2	N	N
44.	Ramesh	32	M	64535	BA	Business	–	–	124	5	110	2.8	138	146/90	+	–	HD/3 months	38	149	205	228	10.2	10.8	N	N
45.	Rajathi	38	F	65175	–	Home maker	–	–	138	5	88	3.5	105	140/100	–	+	Drugs	36	110	110	168	7.4	7.2	N	LVH

46.	Mathivanan	50	M	62252	9th std	Agri	+	+	135	4	140	2.6	106	200/110	+	+	Drugs	38	170	136	235.5	6.5	7.8	N	LVH
47.	Kalaiyarasan	35	M	38420	SSLC	Mechanic	–	+	110	4.8	112	4	130	170/90	+	+	PD/ 2 days	66	148	205	255	11.5	10.8	N	N
48.	Ayyaru	65	M	72015	–	Agri	+	+	128	5	170	5.8	70	190/100	–	+	Drugs	40	128	20	209.6	7.8	8	N	N
49.	Pichai Kannu	60	M	67210	6th std	Agir	+	+	130	4.5	120	3.8	108	138/80	–	+	Drugs	36	66	104	122.8	8.8	9.2	N	N
50.	Anjammal	56	F	71112	–	Home Maker	–	–	110	6	205	12.4	136	170/90	+	+	Drugs	40	128	206	210	8.7	7.8	N	LVH

ACRONYMS

Apo	-	Apolipoprotein
BP	-	Blood Pressure
CC	-	Correlation Coefficient
CKD	-	Chronic Kidney Disease
CVD	-	Cardio Vascular Disease
DM	-	Diabetes Mellitus
ECG	-	Electro Cardiogram
ESRD	-	End Stage Renal Disease
FFA	-	Free Fatty Acid
GFR	-	Glomerular Filteration Rate
HD	-	Hemodialysis
HDL-C	-	High Density Lipoprotein Cholesterol
HTN	-	Hypertension
IDL-C	-	Intermediate Density Lipoprotein Cholesterol
IP No	-	In Patient Number
K^+	-	Potassium
K/DOQI	-	Kidney Disease Outcome Quality Initiative

LCAT	-	Lecithin Cholesterol Acyl Transferase
LDL-C	-	Low Density Lipoprotein Cholesterol
LK	-	Left Kidney
LPL	-	Lipoprotein Lipase
LRP	-	Low Density Lipoprotein Related Protein
LVH	-	Left Ventricular Hypertrophy
MDRD study	-	Modification of Diet in Renal Disease study
Na^+	-	Sodium
OXL-DL	-	Oxidised LDL
PCS	-	Pelvic Calyceal System
PD	-	Peritoneal Dialysis
PMP	-	Per Million Population
RK	-	Right Kidney
Sd LDL	-	Small Dense Low Density Lipoprotein
TC	-	Total Cholesterol
TGL/TG	-	Triglycerides
USG	-	Ultra Sonogram
VLDL-C	-	Very Low Density Lipoprotein Cholesterol

INSTITUTIONAL ETHICAL COMMITTEE
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K.Dis.No.16328 P & D3/Ethies/Dean/GGH/08 Dated: 8.9.2008

Title of the work : "Lipid profile in chronic kidney Disease patients"

Principal Investigator : Dr. A. Vinoth

Department : Institute of Internal Medicine MMC, ch-3.

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 10th September 2008 at 2 P.M in Government General Hospital, Deans, Chamber, Chennai-3.

The members of the Committee, the Secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The principal investigator and their term are directed to adhere the guidelines given below:

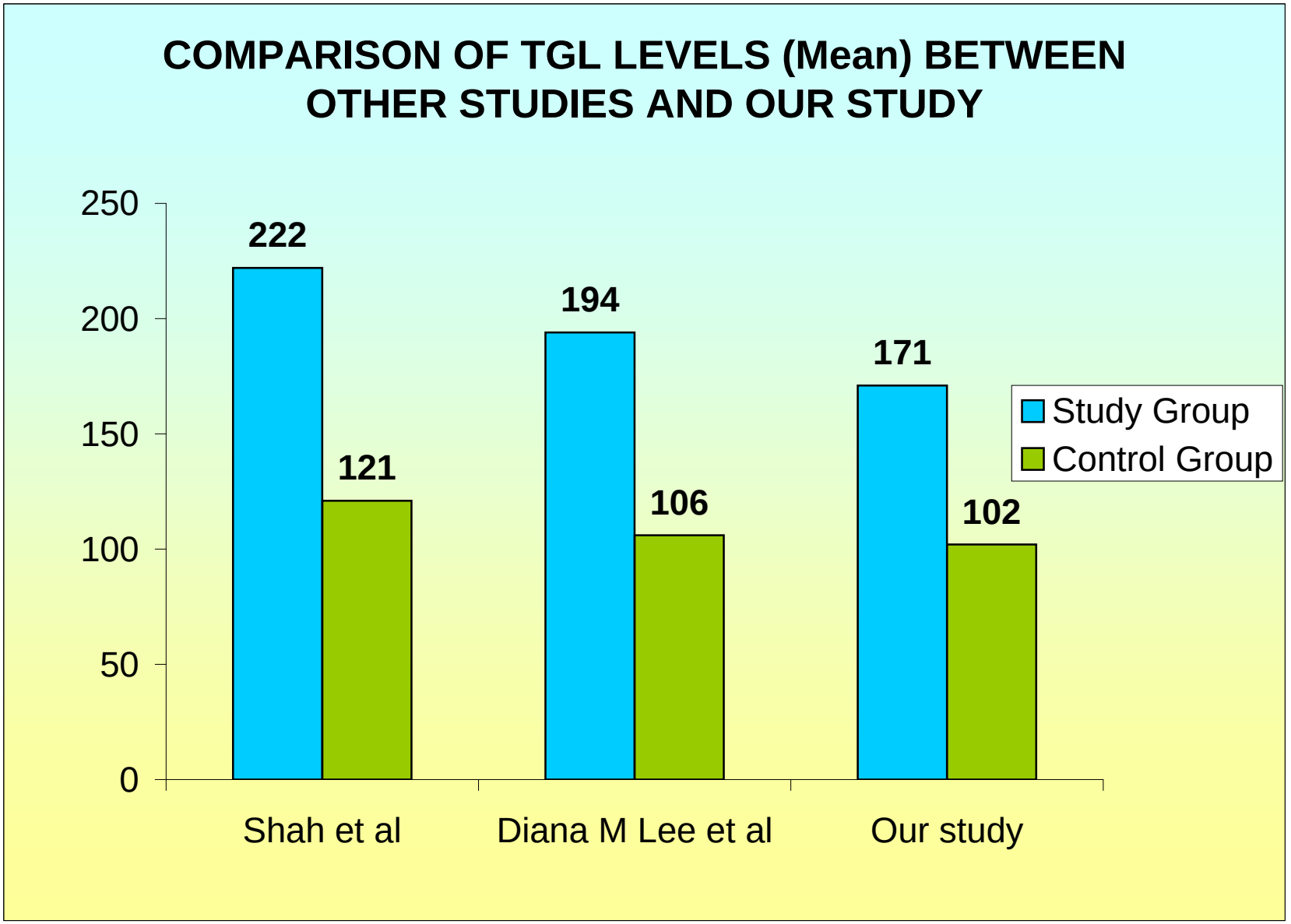
1. You should get detailed informed consent from the patients/participants and maintain confidentiality.
2. You should carry out the work without detrimental to regular activities as well as without extra expenditure to the Institution or Government.
3. You should inform the IEC in case of any change of study procedure, site and investigation or guide.
4. You should not deviate form the area of the work for which I applied for ethical clearance.
5. You should inform the IEC immediately, in case of any adverse events or serious adverse reactions.
6. You should abide to the rules and regulations of the institution(s)
7. You should complete the work within the specific period and if any extension of time is required, you should apply for permission again and do the work.
8. You should submit the summary of the work to the ethical committee on completion of the work.
9. You should not claim funds from the Institution while doing the work or on completion.
10. You should understand that the members of IEC have the right to monitor the work with prior intimation.

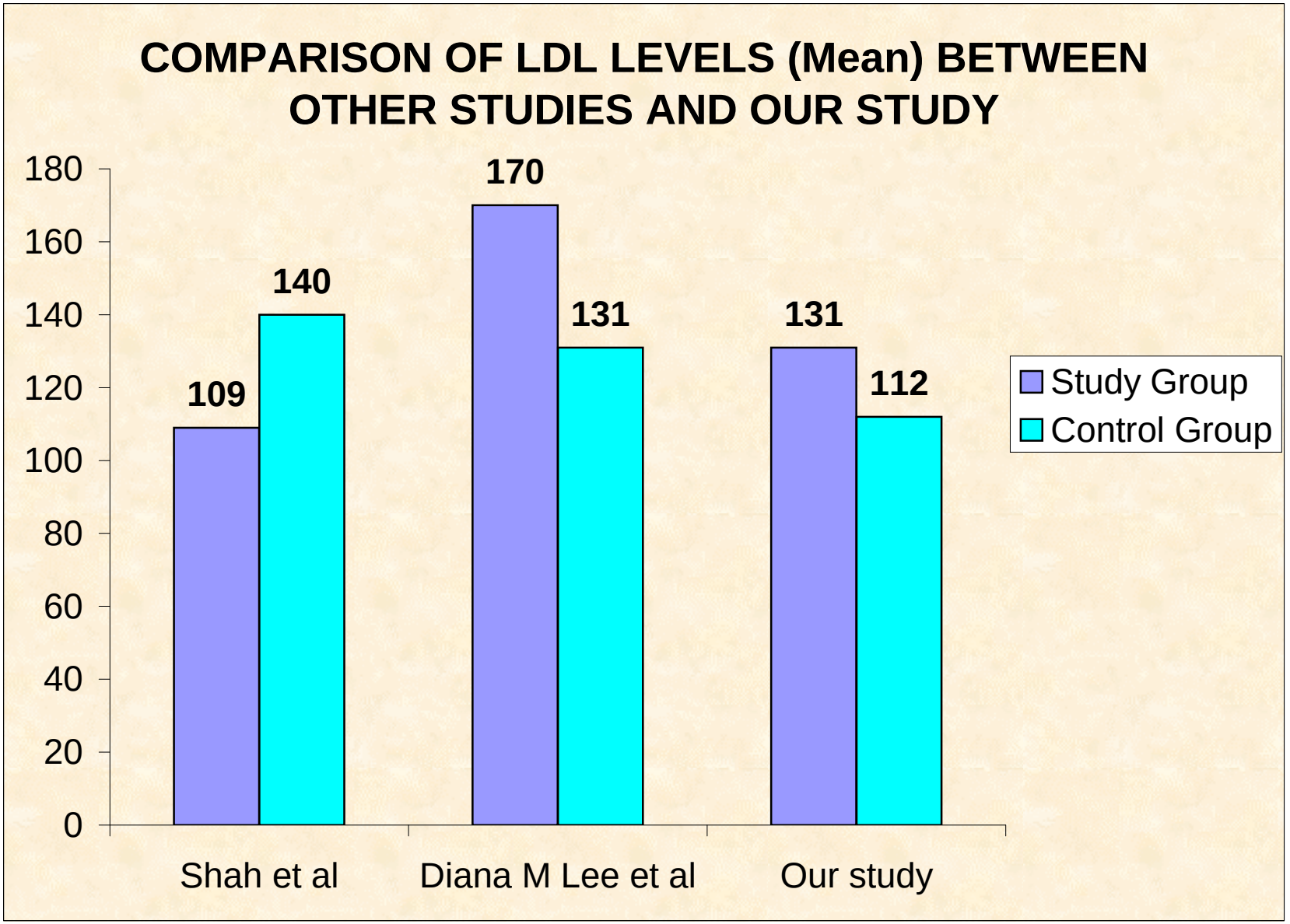
SECRETARY
IEC, GGH,CHENNAI

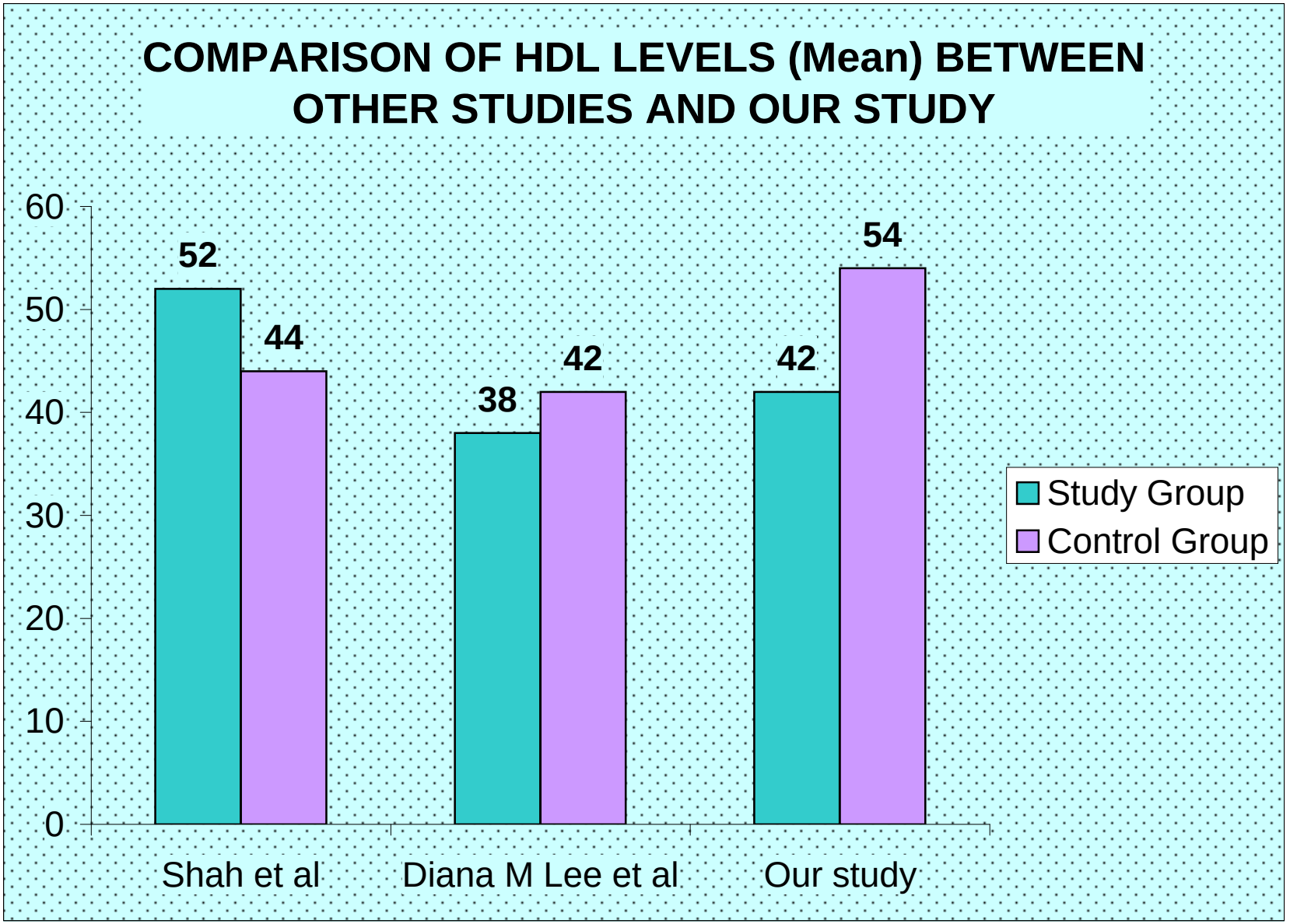
CHAIRMAN
IEC, GGH,CHENNAI

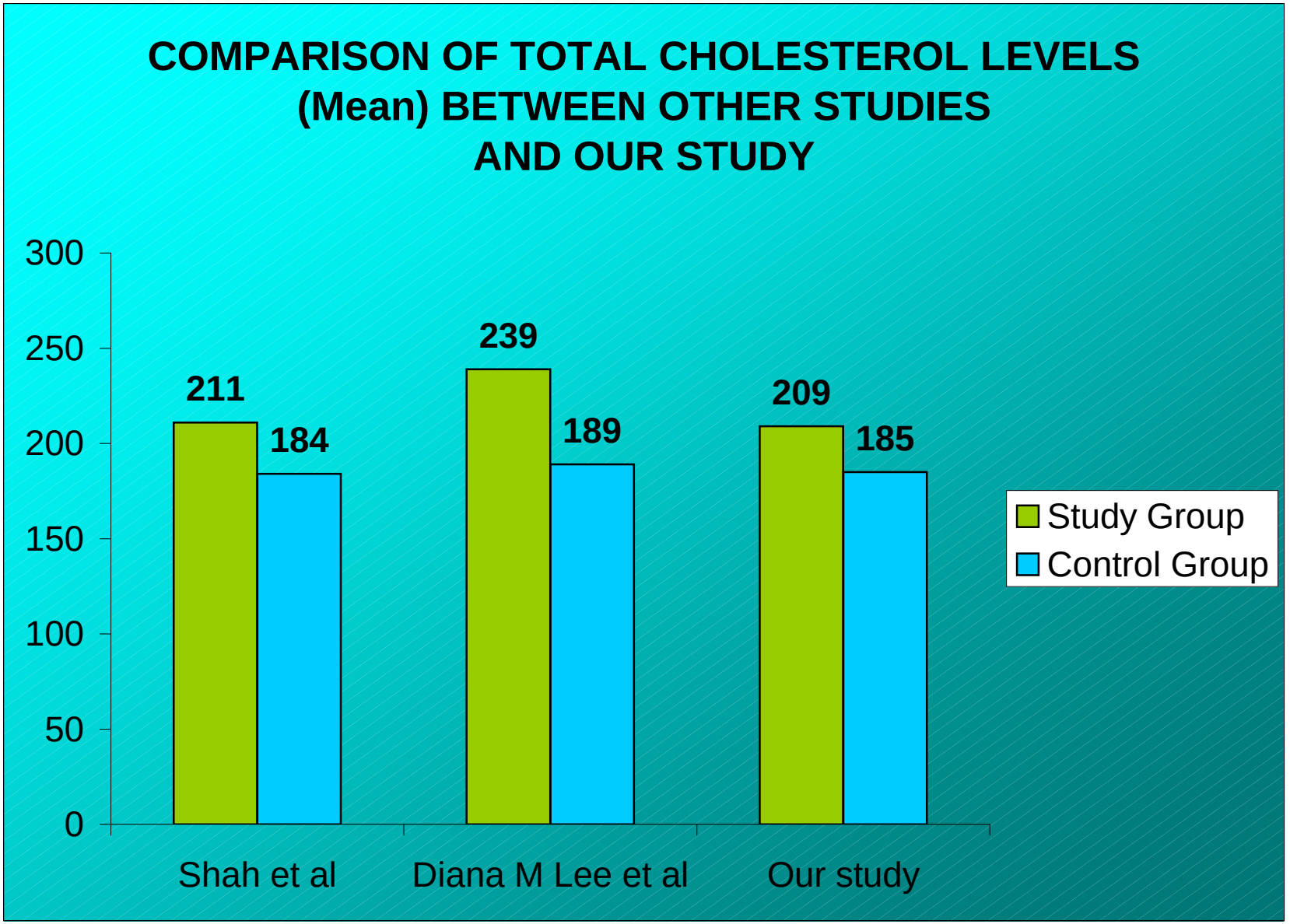
DEAN
GGH &MMC, CHENNAI

Rkm.5.9(2)

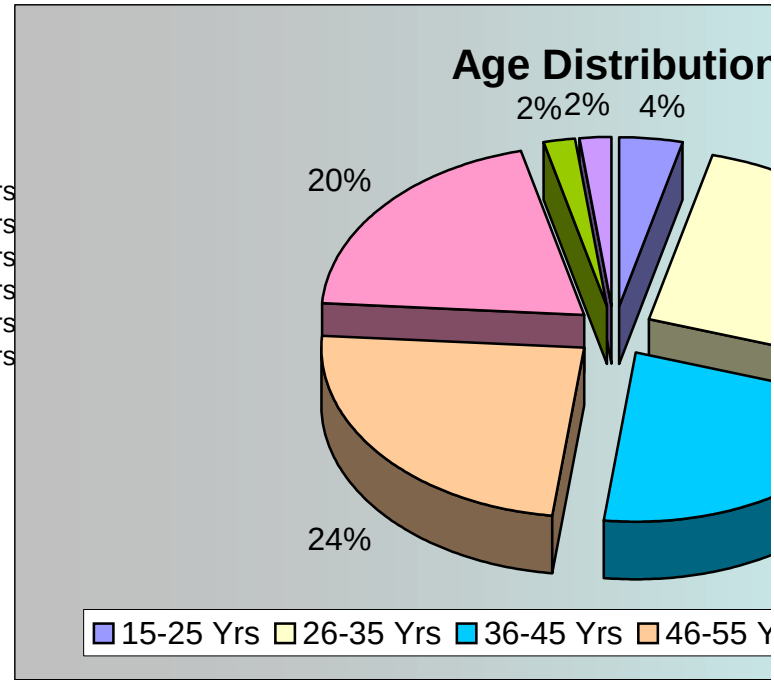

COMPARISON OF TGL LEVELS (Mean) BETWEEN OTHER STUDIES AND OUR STUDY
250
200
150
100
50
0
222
121
194
106
171
102
Study Group
Control Group
Shah et al
Diana M Lee et al
Our study

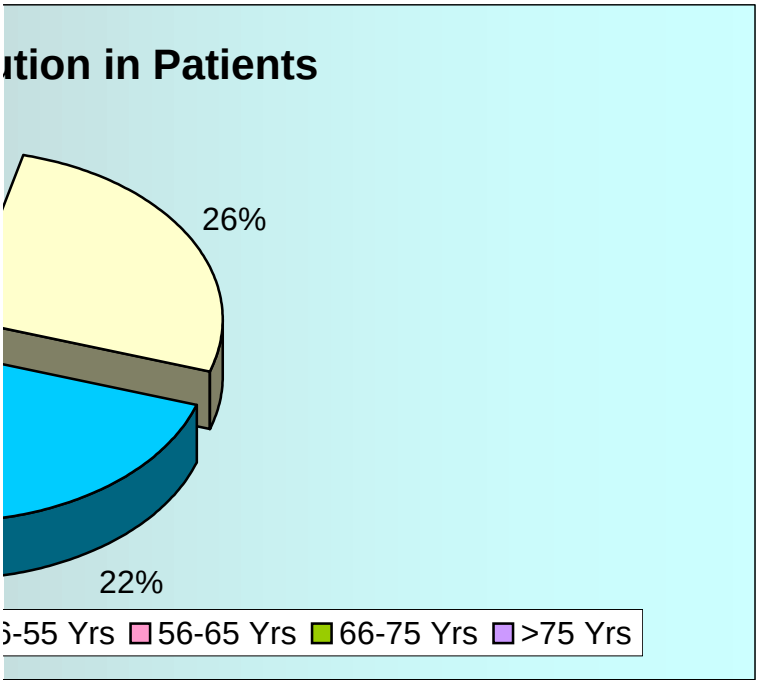

COMPARISON OF LDL LEVELS (Mean) BETWEEN OTHER STUDIES AND OUR STUDY
180
160
140
120
100
80
60
40
20
0
109
140
170
131
131
112
Study Group
Control Group
Shah et al
Diana M Lee et al
Our study

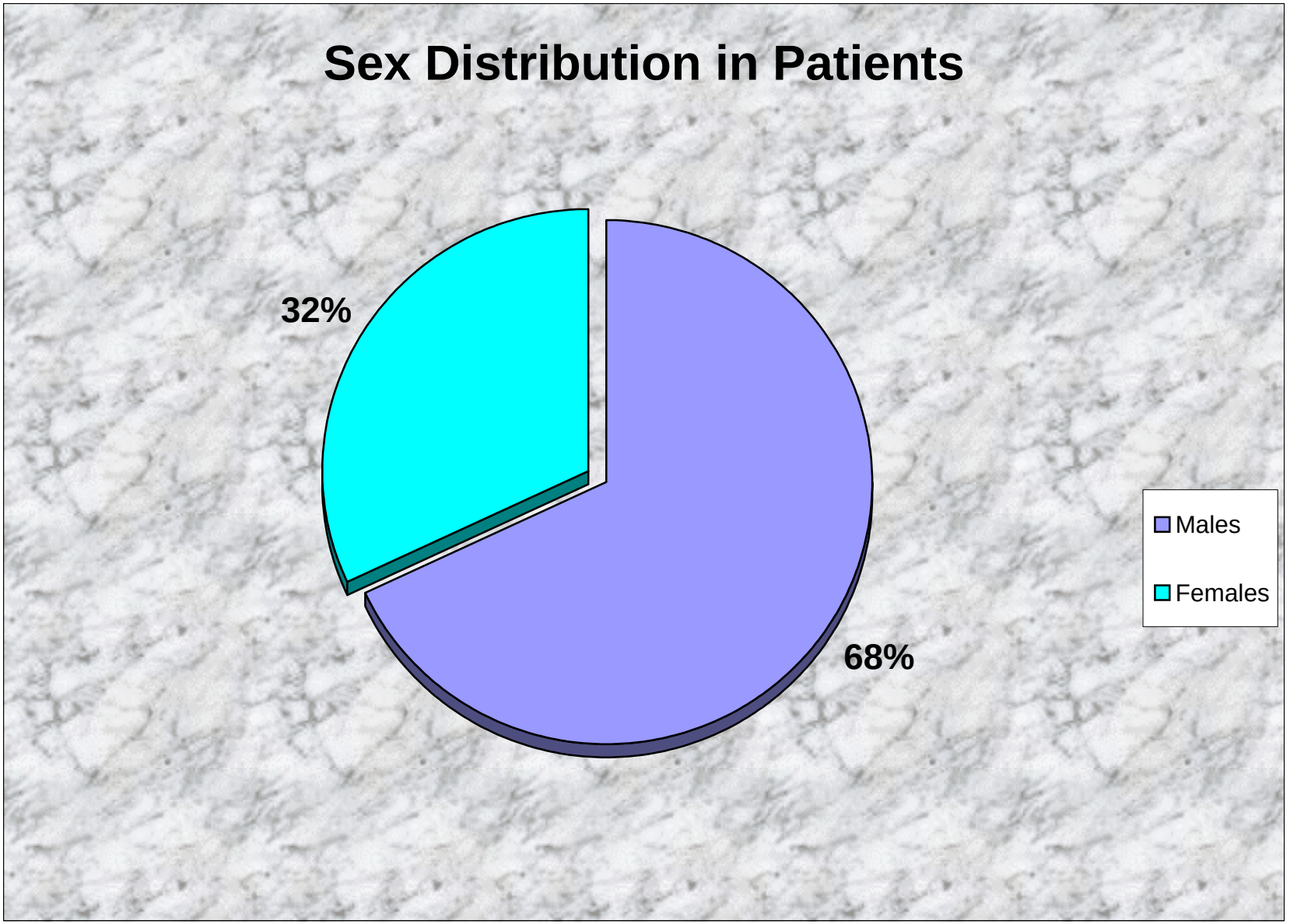

COMPARISON OF HDL LEVELS (Mean) BETWEEN OTHER STUDIES AND OUR STUDY
60
50
40
30
20
10
0
52
44
38
42
42
54
Study Group
Control Group
Shah et al
Diana M Lee et al
Our study

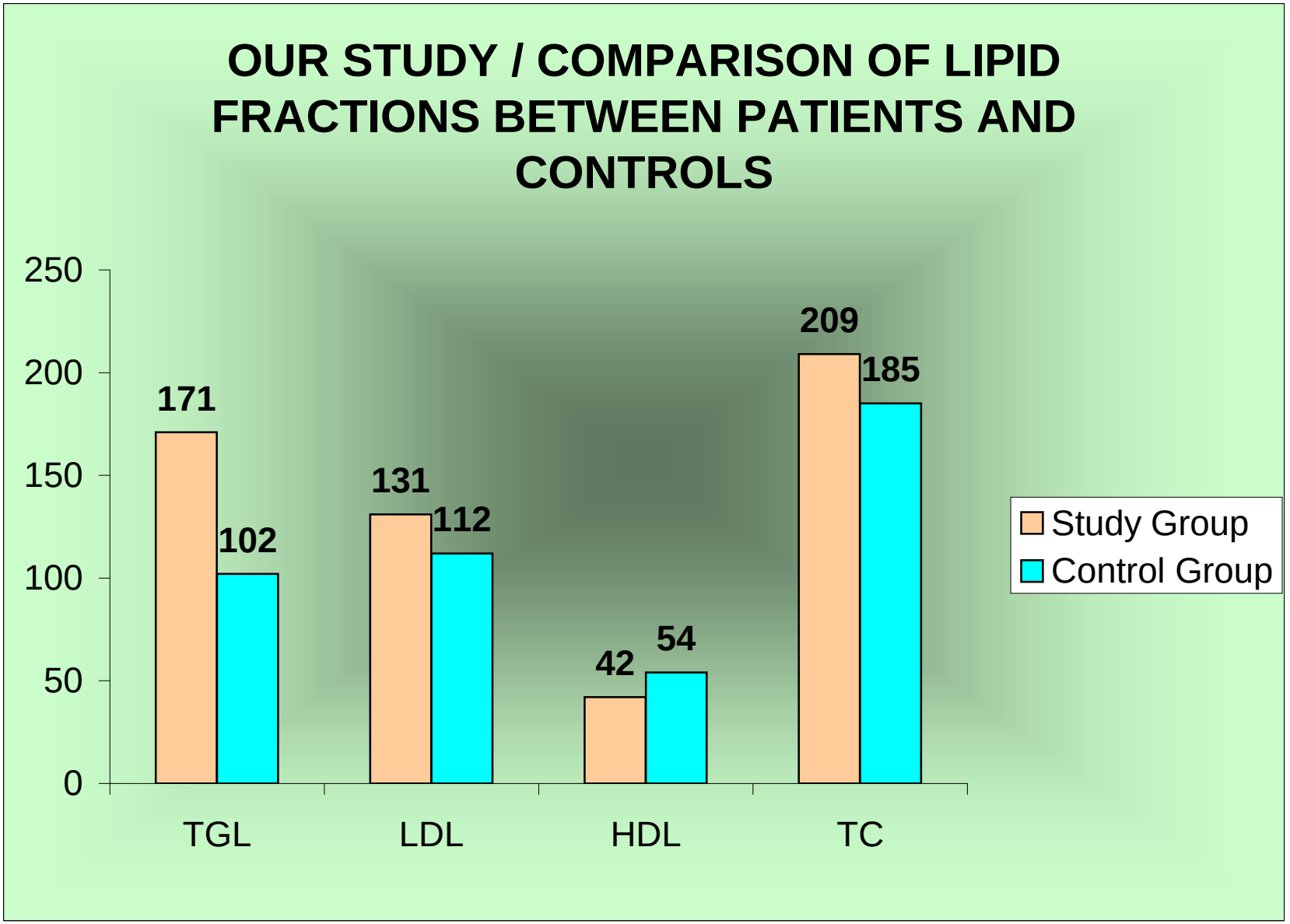

COMPARISON OF TOTAL CHOLESTEROL LEVELS (Mean) BETWEEN OTHER STUDIES AND OUR STUDY
300
250
200
150
100
50
0
211
184
239
189
209
185
Shah et al
Diana M Lee et al
Our study
Study Group
Control Group

15-25 Yrs
26-35 Yrs
36-45 Yrs
46-55 Yrs
56-65 Yrs
66-75 Yrs
>75 Yrs




ıtion in Patients
26%
22%
6-55 Yrs
56-65 Yrs
66-75 Yrs
>75 Yrs


Sex Distribution in Patients
32%
68%
Males
Females


OUR STUDY / COMPARISON OF LIPID FRACTIONS BETWEEN PATIENTS AND CONTROLS
250
200
150
100
50
0
171
102
131
112
42
54
209
185
TGL
LDL
HDL
TC
Study Group
Control Group